



**Agriculture
Canada**

Publication 1706

nutritional muscular dystrophy of young ruminants



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PUBLICATION 1706, available from
Information Services, Agriculture Canada, Ottawa K1A 0C7

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Cat. No. A63-1706/1980E ISBN: 0-662-11167-2
Printed 1980 10M-11:80

Aussi disponible en français

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INTRODUCTION

Many diseases can occur in calves and lambs, but one of the most serious is nutritional muscular dystrophy (NMD). This disease, which can cause heavy losses within a herd, has been studied by several scientists and has been called by various other names, such as white muscle, fish meat, chicken meat, white muscle disease, marble calf, stiffness disease, myopathy, myositis, and muscular dystrophy.

In regions specializing in meat production, this condition can cause serious financial problems for farmers. Therefore, it is important to study the disease and its causes and to establish measures for its prevention and treatment.

This study summarizes the observations published by various investigators and practicing veterinarians. Also some of our own findings and recommendations are included.

SYMPTOMS

Calves

The disease affects calves from 15 days to 4 months old, either when they are still in the barn or within a few hours after they have been put out to pasture for the first time. Often the victims of the disease are the best specimens: well looked after, fast-growing milk-fed calves. The animals afflicted are usually large-rump types with highly developed muscle masses, such as Limousin or Charolais calves. The symptoms appear suddenly and can vary in intensity, because the disease has several forms.

Locomotor signs

This is the most common form of the disease. When the animal is lying down, there are no apparent symptoms. However, the animal may appear somewhat listless and refuse to stand up. Although, on the previous day as soon as the calf was released from its tether, it rushed to its mother to suckle greedily, butting her playfully, it now seems less eager. It no longer frisks about when it is untied to suckle. Soon, this seeming indifference gives way to a stiff gait, which becomes worse. When urged, the calf manages to stand up with considerable difficulty, but tremors may affect its entire body or only certain groups of muscles. The animal stands stiffly on the tips of its hoofs with its back arched and its tail raised as if wanting to urinate. When it moves, its steps are measured, hesitating, and painful as if exhausted from strenuous

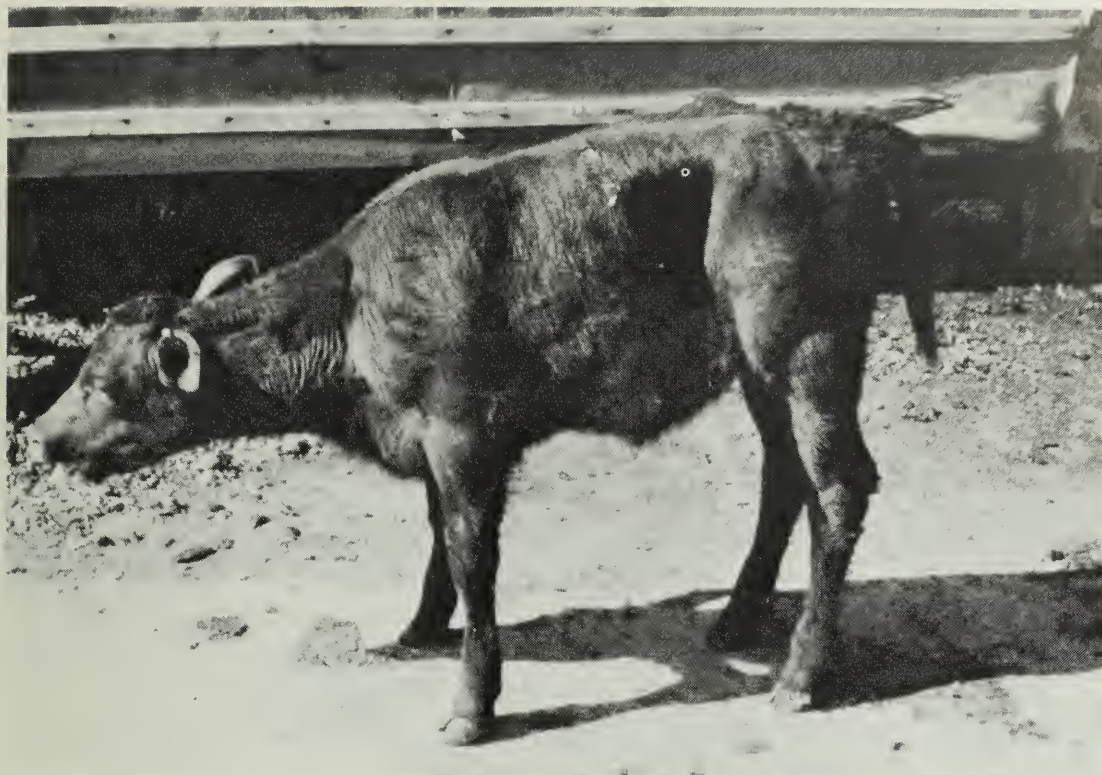


Stiffness in the calf causes it to stand in a urinating position with its back arched and its tail raised. The calf is standing on the tips of its hoofs.

exercise. The stiffness increases. The calf goes back to its stall and falls down on the straw. When you examine the calf, you will find that some of the rump and shoulder muscles feel tense and contracted. Sometimes, the animal can no longer stand, but it still has an appetite. If the mother is brought to it, the calf will suckle from a lying position. The symptoms increase, especially the stiffness, and the calf, stretched out on the litter, cannot respond. The calf needs help to get up and stand under its mother. It might suckle briefly, but it seems quickly exhausted from the effort. During this period, which may last about 10 days, the animal's temperature remains normal (39°C). Later, because the calf cannot feed itself, it dies.

Respiratory signs

This form of the disease occurs suddenly and follows the form with locomotor signs. It is caused by marked degeneration of the respiratory muscles. The animal has trouble breathing, and it breathes in short, quick gasps. All the abdominal muscles are involved. Jerky or convulsive and violent movements increase, and breathing is extremely difficult. This stage is called the heaves.



A calf suffering an attack of shortness of breath has accelerated breathing, fluctuating flanks, and degeneration of the respiratory muscles.

Cardiac form

This is the most serious of all the forms of the disease and is often difficult to distinguish from the respiratory form. Generally, both forms occur at the same time and the animal has great difficulty breathing. The cardiac form may appear first or occur after locomotor disorders. It is the most serious form, because it may result in sudden death. The animal lies on its side and moans periodically. By examining the chest with a stethoscope or by putting your ear to its chest you can hear the heart beating fast and occasionally it seems to beat irregularly or intermittently. Usually, death results from heart failure.

Heifers over one year old

Although the symptoms are the same as those observed in calves, there is one difference: the locomotor signs (gait disorders) occur first, followed by tremors and shortness of breath when the animal tries to stand. Hemoglobinuria, the presence of red blood cells in the urine, continues throughout the

course of the disease. The urine is dark and may become the color of coffee grounds. These symptoms always occur when the animal is put out to pasture, especially if the days are warm.

Lambs

A stiff gait may develop in lambs when they are about 3 weeks old, but it occurs also in younger and older animals (up to 2 months). In the early stages, the animal's hindquarters seem to be partly paralyzed, because the hind limbs are the first to be involved and they have trouble following the forelimbs. The lamb soon lies down. It can raise its head, neck, and forequarters, but the lifeless rump remains on the ground. The animal can no longer follow its mother and it stays on the straw. It does not seem to suffer, and will suckle if the mother is brought to it. Nevertheless, the lamb loses weight and dies within two or three weeks, when weight loss is extreme. The respiratory and cardiac forms of the disease are less common in lambs, but the symptoms are the same as in calves. Usually both forms occur together, but if only the heart is involved, it beats very fast and breathing is accelerated. The animal cannot stand. It dies within a day or two.

LESIONS

Sores form at all bony protrusions as a result of extended periods of lying. Sometimes, bronchial pneumonia develops. Generally when a calf with respiratory distress is slaughtered because of consumption, these lesions are easily seen.

The animal's lungs enter a state of asphyxia with edema and spumescence (bloody froth) in the bronchial tubes and trachea. The heart has putty-colored streaks, which correspond to the degenerate portions of the muscle. These lesions tend to calcify quickly, which explains their whitish color. The lesions involve primarily the muscle mass. Therefore, the color of the muscle ranges from pale pink to white, often with whitish veins. For this reason, calves with the disease are referred to as marble calves. At times, the muscle is completely discolored. The muscles most often affected are the shoulder and thigh muscles. These muscles work hardest and have the most blood vessels. There are also a large number of lesions on the diaphragm and on certain respiratory muscles. These muscles are less firm to touch than normal muscles. A large amount of fluid oozes from the meat of diseased calves and the flesh is softer than that of healthy calves. The appearance of the lesions and the consistency of the meat do not change in cold storage. Because the flesh varies from pale pink to white, affected carcasses are referred to as white meat, chicken meat, or fish flesh.



Degenerate muscles resemble fish flesh.



Degeneration of the heart is shown by putty-colored lines with calcium deposits.

Sometimes when symptom-free calves from farms where the disease is rampant are slaughtered, their flesh appears abnormally pale. Do not eat this meat. Affected meat requires more cooking and yields a considerable amount of liquid, which gives the meat a bland taste. The lesions that have just been described produce the main external signs of the disease, which include a stiff and unsteady gait, respiratory distress, paralysis (often of the hind limbs), and, occasionally, sudden death.

CONSEQUENCES

Both the economic and medical results of NMD are serious.

Economic

The economic outlook is serious. Losses are hard to assess accurately, because many dead animals are buried by their owners and the deaths are not recorded. The survivors are without value during their convalescence, which is always slow. Even after the animals have recovered, they never regain their original vigor. Their growth, retarded by the disease, continues thereafter at a slower than normal pace.

Medical

The medical consequences are hard to determine. The effects of the disease are less serious when calves with only locomotor disorders and no signs of heart or respiratory muscle involvement are treated rapidly. If the animal survives 8 days, it probably will live, but recovery is always slow.

Muscular dystrophy is serious because of ignorance about the disease and the lack of care taken when the first symptoms of it appear.

DEVELOPMENT OF THE DISEASE

NMD in young ruminants developed at the time when intensive herd management evolved and progress was made in the selection and feeding of farm livestock. In general, the victims are usually animals from meat breeds. The disease usually occurs in spring, when the animals are put out to pasture. The percentage of diseased calves or lambs varies each year. A larger number of cases have been reported in animals whose mothers were fed during the winter on hay harvested during rainy periods.

When the young animals are first put out to pasture after the inactivity of being in a stall, the sudden, continual movement may also make them susceptible to the disease. The young calf gambols, runs, leaps, frolics, gets out of breath, and, as a result, makes a considerable muscular effort. The animal's heart must also pump harder to supply oxygen to the muscles. Most cases of NMD occur during this time when the animals are first put out to pasture.

CONDITIONS THAT TRIGGER THE DISEASE

Nutritional disorders play a key role in triggering NMD. For a long time, it was believed that vitamin E was important only for reproduction. However, evidence has recently been produced to show that a deficiency or absence of this vitamin results in severe muscular disorders. Vitamin E is found in large amounts in meadow grass (80–150 mg/kg of dry matter); hay contains only small amounts (10–20 mg/kg).

In winter, when cows are fed a dry diet, there is little source of vitamin E, and therefore its level in the milk is low. Thus, a milk-only diet, which is what young ruminants receive, is deficient in vitamin E. This is particularly true toward the end of winter, when the young animals receive poor quality milk.

The disease occurs more commonly in some regions than in others, but we do not know why.

Over the last few years, there has been increasing evidence that the disease occurs more often in regions with granite soils, which are poor in selenium. In these areas, the disease is common.

Analysis of forage from farms where NMD is prevalent has shown low levels of selenium (less than 0.1 ppm). However, the disease has never been reported from farms where this trace metal is found in higher concentrations.

Therefore, muscular dystrophy is a nutritional disease caused by a deficiency of vitamin E or selenium in the diet of calves and lambs.

PREVENTION

Because movement plays an important role in triggering NMD, calves wintering in the barn must have some daily exercise. By this means, when they are put out to pasture, the transition is not too sudden from immobility (which at times is total) in the barn to continuous and often rough exercise, which the animals indulge in when they are let loose in the open. It also has been observed that NMD is more common in animals put out to pasture in sunny weather. Thus by putting the animals out to pasture in small groups for short periods under overcast skies onset of the disease may be prevented.

Because NMD is a nutritional disease, it recurs every year at the same time on the same farms, causing heavy losses. Therefore, it is feasible to try

to prevent it by dietary measures. Studies have shown that calves of cows fed during the winter with first-cut silage were far less subject to NMD than calves of cows fed on hay, both feeds having been produced in a region where the disease in calves was endemic. Although silage made from young herbage is low in selenium, it is rich in vitamin E, which explains its beneficial effect. As a preventive measure, vitamin E is recommended for calves in doses of 150 mg per day from the time of their birth. However, instead of daily doses, it is preferable to give a massive dose of 3000 IU of vitamin E by injection to calves that are threatened by the disease, the day after their birth. The dose should be repeated 1 month later, and 48 hours after the animals are put out to pasture.

The administration of selenium is also effective. In calves, the recommended oral dose is 5 mg every 2 weeks up to the age of 3 months. Half that dose is recommended for lambs, owing to their smaller size. An injection is even more effective. The dose for a calf is about 3 mg selenium per 50 kg of liveweight. Give two more injections at 30-day intervals. The dose for a lamb is 1 mg selenium, intramuscularly, repeated twice. Selenium may also be used as an additive in minerals, in doses of 0.3 ppm in dry feed for suckling animals and in milk replacers for calves up to 8 weeks old. Also, 25 ppm selenium may be added to trace element-enriched salt for ewes, breeding cows, or heifers. Never mix selenium with feed for milk cows or heifers producing or likely to produce milk for humans.

NMD has never been diagnosed in animals in which the feed dry matter contained more than 0.1 ppm selenium. On farms where the disease is thought to exist, be sure to analyze the forage produced on the farm to determine the selenium content. It is then possible to predict cases of NMD by identifying areas where the selenium content is low. Selenium levels in the forage can be determined at an agricultural school or at a provincial veterinary laboratory that specializes in analyzing minerals contained in feeds.

TREATMENT

Most important, the sick animal must have rest, because its muscles have been damaged and they must be allowed to recover. Rest is essential for the success of any medical treatment. Untreated cases, where the animal recovered from rest alone, have often been reported. Take the mother to the young animal and let the sick animal feed in a lying position.

Vitamin E combined with selenium usually gives good results. An intramuscular injection has the advantage of acting more quickly. A preparation containing 6 mg selenium and 300 IU vitamin E per 50 kg of liveweight is recommended for calves. If the calf does not respond to this treatment, another injection can be given a few days later. For lambs, use one-quarter of the dose.

Occasionally this treatment is not effective; failures are due solely to the lack of absorption of vitamin E. Therefore, it is best to administer the vitamin as early as possible.

CONCLUSIONS

Evidence indicates that muscular dystrophy is caused by complex nutritional disorders. The cause is closely bound up with the metabolism of young ruminants and their capability of absorbing selenium and vitamin E. Despite the success of a combined vitamin E-selenium treatment, the disease remains a serious problem, because it is related to the feed given to the mothers during the winter while they are still nursing their young. Therefore, the daily food intake of the calves should be supplemented, according to recommendations, with a preventive treatment of vitamin E and selenium. Also, do not allow the young animals to remain completely immobile in the barn. They should be exercised daily and gradually prepared for the time when they will be put out to pasture. Otherwise, the transition from immobility to frisky activity could be fatal.

Postmortem examination of the muscles of a victim of NMD reveals the degree of involvement of the muscles with the disease and provides an explanation for the clinical signs (stiffness and respiratory distress) and sudden death often resulting from this disease.

NMD often causes heavy losses to breeders in regions producing beef calves and especially to producers of veal calves.

CONVERSION FACTORS

Metric units	Approximate conversion factors	Results in:
LINEAR		
millimetre (mm)	x 0.04	inch
centimetre (cm)	x 0.39	inch
metre (m)	x 3.28	feet
kilometre (km)	x 0.62	mile
AREA		
square centimetre (cm ²)	x 0.15	square inch
square metre (m ²)	x 1.2	square yard
square kilometre (km ²)	x 0.39	square mile
hectare (ha)	x 2.5	acres
VOLUME		
cubic centimetre (cm ³)	x 0.06	cubic inch
cubic metre (m ³)	x 35.31	cubic feet
	x 1.31	cubic yard
CAPACITY		
litre (L)	x 0.035	cubic feet
hectolitre (hL)	x 22	gallons
	x 2.5	bushels
WEIGHT		
gram (g)	x 0.04	oz avdp
kilogram (kg)	x 2.2	lb avdp
tonne (t)	x 1.1	short ton
AGRICULTURAL		
litres per hectare (L/ha)	x 0.089	gallons per acre
	x 0.357	quarts per acre
	x 0.71	pints per acre
millilitres per hectare (mL/ha)	x 0.014	fl. oz per acre
tonnes per hectare (t/ha)	x 0.45	tons per acre
kilograms per hectare (kg/ha)	x 0.89	lb per acre
grams per hectare (g/ha)	x 0.014	oz avdp per acre
plants per hectare (plants/ha)	x 0.405	plants per acre

